dr Chetty's Covid Treatment Part 6

66-84 minutes

Beginning of the translation of the interview by Dr. Philip McMillan with Dr. Shankara Chetty, 4 Dec 2021

dr Phillip McMillan: Hello [00:01:00] and good afternoon, good morning, good evening, wherever in the world you are, thank you for joining me on this other topic about COVID 19 in South Africa and the Omicron update . There isn't much to report as there is still a lot of research to be done, [00:01:30] but we have Dr. Shankara Chetty there. I spoke to him for the first time in April of this year and was blown away by his knowledge and what he had achieved at that point: over 4000 COVID patients treated and no deaths. He's now over 7,000. It's almost unbelievable and there's so much to learn from him just by listening to him and [00:02:00] hearing from him. And so it is with great pleasure that I welcome Dr. Shankara Chetty here and Shankara if I had a big round of applause for the sound

dr Shankara Chetty: [00:02:13] Thanks for letting me be back, Philip.

Philip: [00:02:14] Excellent. Excellent. How are you? As I said before, I feel like this video has been viewed over two hundred and eighty thousand [00:02:30] times since our interview in April. They have been called all over the world to help each other in different countries. We'll get to that in a moment. But I think the weirdest thing I've seen, and I've observed it in a lot of the comments, is that a lot of people just don't believe it. They say that's not possible. What do you make of it?

Shankara:[00:02:55] Oh, that's happened to me too, Philip. I think the [00:03:00] world is too fixated on data and randomized clinical trials and the rest. I had a discussion with Chris Newton and I said to him, you know, we're a bit off track as science. The pharmaceutical industry has introduced randomized clinical trials and you usually have this bell curve when you're testing a new drug against society and we focus on the middle of that bell [00:03:30] because we want to know what that drug is causes. But we're dealing with a pandemic here, and we've gleaned a wealth of knowledge before we even got to this virus. So if we deal with that, we still get a bell-shaped curve. But the center of the bell is well known. That's what we expect from a respiratory virus. So what helps us understand is the tails of that bell-shaped curve, the unusual patients, the things we didn't expect, and that's what brings new scientific discoveries. So I [00:04:00] think doctors need to go back to discussing the unusual cases that are discussed in the doctor's room at the end of the day, and we're going to find some breakthroughs in science. So I think, Philip, the simplicity of what I've discovered is fascinating at times. It can't be that easy, can it? the unusual patients, the things we didn't expect, and that's what brings new scientific discoveries. So I [00:04:00] think doctors need to go back to discussing the unusual cases that are discussed in the doctor's room at the end of the day, and we're going to find some breakthroughs in science. So I think, Philip, the simplicity of what I've discovered is fascinating at times. It can't be that easy, can it? the unusual patients, the things we didn't expect, and that's what brings new scientific discoveries. So I [00:04:00] think doctors need to go back to discussing the unusual cases that are discussed in the doctor's room at the end of the day, and we're going to find some breakthroughs in science. So I think, Philip, the simplicity of what I've discovered is fascinating at times. It can't be that easy, can it? So I think, Philip, the simplicity of what I've discovered is fascinating at times. It can't be that easy, can it? So I think, Philip, the simplicity of what I've discovered is fascinating at times. It can't be that easy, can it?

Philip: [00:04:21] That's part of the problem, because people think it can't be that easy. We'll discuss that in a bit more detail. [00:04:30] But I want to take a little time, Shankara, and I want people to understand the principles of simplicity in what you've done. So here is a picture of you with your team. And it's only two nurses, isn't it?

Shankara: [00:04:47] Yes, there are two nurses. I use the third when we're busy in the middle of the waves. Unfortunately, she's in high school. She takes control of my front gate. So I think [00:05:00] that she will come back with Omicron this holiday season to support me.

Philip: [00:05:04] Mm-hmm. And for those who want to get an overview, here's another image that shows how you set your spacing. you have your tents You said someone tried to steal your tent, is that correct?

Shankara: [00:05:19] Yes, yes. Yes, I'll have to get a new one, especially for Omicron, the little thing on the side. Sunny South Africa but I've heard sunshine is good for Covid so I'm considering [00:05:30] getting the tent to leave them in the sun for a bit.

Philip: [00:05:33] And here's a picture of the building that's across the street. I'll retrieve it right here. Across the street you can see your tent on the right. What is this tent for?

Shankara: [00:05:56] Outside, Philip, I need to [00:06:00] distinguish between COVID positive and COVID suspect. This area is therefore intended for COVID-positive patients. If you look around my garden in the background, you can see people staying at the boundaries of my property to socialize. The other tent, or the main tent, is for investigation and on the

side they have a small pagoda. This is for COVID suspects. And those who are there for other reasons are allowed to enter the practice themselves.

Philip: [00:06:28] Wonderful, wonderful, wonderful. [00:06:30] So I'll tell you something Shankara, so only for the people that you may not have heard before. Let's briefly recap what happened in April or May 2020 that prompted you to take this unusual approach with no fatalities to date.

Shankara: [00:06:54] Yes, I have had patients who were hospitalized where [00:07:00] either my treatment was discontinued and their condition worsened, or patients who were hospitalized where other treatments caused her death. But in terms of my protocol and outpatient treatment, even though I had critically ill patients, everyone who stayed with me has recovered.

Philip: [00:07:19] And quite sick patients. Tell us what happened in the early stages of the pandemic. Because I want to use this approach and this analysis [00:07:30] to take a closer look at Omicron. What made you look at COVID 19 differently back then?

Shankara:[00:07:41] Philip, speaking of the virus, I know that humanity has encountered coronavirus before and I understood this type of disease, so I had an idea of the nature of the disease, distribution, epidemiology and the like. And that's how I saw it before it [00:08:00] came to South Africa. And then I want you to understand the presentation, the clinical picture. I had to figure out how to treat this disease. So I looked at what was coming out of Italy back then. Much has been reported about dyspnea. But remember, these are clinical pictures of patients in the hospital, and these are seriously ill patients. So we knew about the lowering of the oxygen saturation of the blood, we knew about the “happy hypoxia” [00:08:30]. So there were certain clinical signs and symptoms that were unusual. We knew about the loss of smell and taste. But we also heard from people who dropped dead on the street. That was very unusual. It was almost like not knowing how sick you were, and suddenly the illness caught you by surprise. So I knew I was going to be dealing with something unusual, and I needed to understand what was unusual. So the world called [00:09:00] this a COVID pneumonia. And I thought that education and understanding will come from examining the patient from the day they develop the disease until they present to the hospital. I felt that was not the case with the pandemic itself. So I decided that I had to resort to my own education. I decided to pitch a tent. I have decided to educate my community that there is nothing to fear, we will [00:09:30] be with them from the start. I took the necessary steps to set up the tent so I could schedule the patients. Ventilation and sunlight have always been the best disinfectants you could find. And so it was an easy task for me to make sure that I was available to the patients. I needed to understand what exactly was going on. we will [00:09:30] accompany them from the beginning. I took the necessary steps to set up the tent so I could schedule the patients. Ventilation and sunlight have always been the best disinfectants you could find. And so it was an easy task for me to make sure that I was available to the patients. I needed to understand what exactly was going on. we will [00:09:30] accompany them from the beginning. I took the necessary steps to set up the tent so I could schedule the patients. Ventilation and sunlight have always been the best disinfectants you could find. And so it was an easy task for me to make sure that I was available to the patients. I needed to understand what exactly was going on.And of course I didn't want to draw the attention of the authorities, so I moved out of my [00:10:00] house and into another apartment... I normally live in the same building where my practice is located. To protect my family, I moved to another apartment to isolate myself. I spent the first four months examining patients and living in isolation. But I have taken the time to examine each patient and try to understand how this disease progresses. In viral infections, the course is usually very typical. Unlike a bacterial infection, which can last two days or two months. Viral infections have a finite [00:10:30] time in their host, like measles, chicken pox, and all these types of viruses that we deal with. So I want that you understand how this virus evolved to the point where dyspnea and hospitalization occurred. So that was the goal I was pursuing with this work.

Philip: [00:10:50] And that's an interesting point, you know, Shankara, you said something that really got me thinking. They went to the front and [00:11:00] looked after the patients and tried to understand the clinical picture. Why isn't this kind of observational work respected in medicine anymore?

Shankara:[00:11:19] I think Philip, medicine has become too commercial, dictated by drug companies, dictated by algorithms, dictated by protocols. I think in our interview [00:11:30] I mentioned that I treat patients and not blood test results. That seems to have gone down well with older doctors. The younger ones are taught to look at blood test results, and you know. I think medicine has moved away from what it was supposed to be. Clinical examination of the patient, understanding. I think that's what the doctors should do. Look at the patient and understand the symptoms. There is

[00:12:00] a wealth of knowledge that one learns when examining a patient. You know, in my practice I set it up like this that I can watch a patient come into my office. I don't sit behind a desk. I could never speak to my client from there. So I want patients to feel comfortable so they can tell me all their problems. And of course if someone comes in with a limp and I'm sitting behind the desk and walking or coming in while they're sitting, I wouldn't know they were limping. So I think the examination [00:12:30] of a patient begins before they're even greeted. And of course the way a patient greets you, shakes your hand, makes eye contact. All these little nuances give you a lot of information about the patient you are dealing with. And no blood test or exam, no matter how extensive, will give you that kind of knowledge. So when it comes down to the patient themselves, this is the path you need to take. No one is like the other, Philip.

Philip: [00:12:57] Yes,

Shankara: [00:12:59] I think we have to take that into account [00:13:00].

Philip: [00:13:01] Yes. Well, absolutely right. When I first spoke to you, I realized that you have a wealth of experience because you see patients very, very early. Because the doctors in most other countries refused to see the patients early. You would just end up in the hospital. So you missed the critical phase where you could have observed the patients at a very early stage of the disease and figured out what on earth [00:13:30] was going on. And in doing so, you stumbled upon a very important observation: the eighth day. Can you just explain that to people?

Shankara:[00:13:45] When the first cases of COVID started to appear, I already had an idea of what I might be dealing with. The progression of the disease from what I had seen in Italy. You [00:14:00] have seen people where the disease progressed very quickly. And at the other end of the scale, there were people who said it was just common flu and they didn't get all the hype. So there was this mix of cases that you saw and this spread of the disease. So I knew I had to document this carefully. And that's how I talked to every patient. I explained to them that I must be informed immediately if their symptoms worsen. I wanted to ensure a good clinical recovery and a speedy [00:14:30] recovery and to investigate the dyspnea. Understand, what caused this dyspnea (shortness of breath). And initially, the first patients I saw had a typical flu-like viral illness that showed signs of remission after three or four days. They recovered easily. And then I had the first patient who came in breathless. Of course I had to think about what to do at this point. We knew that this is a steroid-responsive disease [00:15:00] and that this is the ideal time to start steroid treatment. It was obvious that something had changed that day. So I put the patient on steroids and it took a day or two for him to start recovering. After about three days of steroids, they showed good signs of recovery. And then the second patient came in. Then, with the third or fourth patient, I began to see a pattern: each and every one of those patients came to me exactly a week after the first symptoms appeared. So I [00:15:30] went back to my files and checked them again and they were all the same. All patients who developed shortness of breath appeared to develop shortness of breath as early as one week after their symptoms began. Of course I asked all these patients how they got to this point. They were all fine the day before. There was no progression. Pneumonia is a progressive disease. It starts with a sore throat, progresses to bronchitis and [00:16:00] then you get short of breath and you have pneumonia. You're not completely healthy, you wake up one morning and you're out of breath, and that is then considered pneumonia. So the disease itself did not fit the diagnoses I heard from around the world, and the clinical examinations of these patients were unusual. Pneumonia occurs in seriously ill patients. These patients were not seriously ill. They were exhausted. They were breathless but otherwise fine. [00:16:30]You would never say that this is a patient with pneumonia. When these patients were examined, it was found that they had good access to the lungs. The sound of your breath sounds normal. Unlike pneumonia, I couldn't get them to crepidate (rattle). The only respiratory symptom I noticed was that they complained of not being able to breathe deeply. That's all. And I've noticed that her saturation has decreased. So this is not the [00:17:00] typical clinical picture you would see with pneumonia. And of course it clarified that I was dealing with a biphasic disease. On the eighth day something had changed. All had shown signs of improvement for the first seven days and then suddenly worsened on the eighth day. So I knew I was dealing with a biphasic disease. It didn't seem to have linearity. Of course, the second part had nothing to do with the first. There was no correlation [00:17:30] between the two phases. I've had patients who get a sore throat recover within two days, perfectly healthy for the rest of the week and exactly one week later suddenly had shortness of breath. That was strange. This is not the typical course of pneumonia. So it became clear to me very early on that I was dealing with a different pathology. And so I had to take the time to find out what this pathology actually is.

Philip: [00:17:59] All [00:18:00] I can tell you is that every time I listen to you and you talk about it, I get goosebumps when I get to the basics of the Medicine think that's how medicine should be. Observe, reflect, apply, observe, reflect, apply. So

this led to you observing day 8, and technically observing day 8 with the administration of antihistamines and steroids [00:18:30] resulted in you being able to save every single patient you came across.

Shankara:[00:18:38] Yes, Philip, you see. You see, as a doctor, every patient that comes to us is a therapeutic attempt. I don't know if the medication will bring any improvement. I rely on my ability to diagnose. And, of course, the diagnosis is usually a differential diagnosis and treated with the most appropriate therapy. And [00:19:00] if you fail, then move on to the second best solution and keep exploring. Thus, each patient undergoes an attempt at therapy, and the success of the attempted therapy is determined by two things: speed of symptom resolution and complete recovery. So a speedy recovery is crucial. It makes no sense that I give you a paracetamol and wait five days for your headache to go away, and then think that the paracetamol did something for you. There must be a rapid response [00:19:30] to treatment. And in any treatment that is a therapeutic attempt, rapid response, rapid recovery, will dictate or augment the underlying mechanism. So that's the way I practice medicine. And so I started the steroids. I understood that I was dealing with this variety of reactions in terms of severity that day. So the only thing that seemed to fit was an immune-mediated reaction [00:20:00] and not a worsening viral infection. And so I figured if I'm dealing with an immune response, looks like an allergen or some kind of hypersensitivity, then the administration of an antihistamine would be the logical next step. I think the fifth patient out of six who came to see me had an antihistamine in addition to the steroid and then compared the speed of healing. And while it took three days to get better with the steroid, with the addition [00:20:30] of an antihistamine, the patient was fine the very next day. And so it accelerated recovery. That made me aware that I was dealing with hypersensitivity. And from that point on, the science follows the training. We know what causes hypersensitivity, the release of mediators and what to do to eliminate them. And of course I focused on what could be the likely allergen, the culprit, that triggers this reaction. At this point [00:21:00] every patient I have treated has had the same success. So I think the focus has now shifted from treating COVID to understanding what caused it.

Philip: [00:21:10] Someone asked this question, which I know is important: what steroid were you using? Because dexamethasone is used all over the world, but not you. Why is that?

Shankara:[00:21:24] Look, dexamethasone is an injectable solution, and being an outpatient facility, [00:21:30] I don't have the facilities to put IVs and monitor all these patients. So I knew I needed a steroid, and of course I'm very knowledgeable about prednisone. It's a cheap drug and I've been using it for many years. So I thought, well, let's start with that. If I had difficulties, then there would be a reason to change. And of course some of my patients are from a very poor demographic and prednisone is the cheapest steroid you can find there. And so I [00:22:00] decided to start prednisone. And in the recoveries I've had, I haven't found a reason to really change that. Yes, in the patients who could afford it, I switched to methylprednisolone because it just deposits better in the lungs and that's where the pathology is. And of course a little less problems with hypoglycemia. I've had patients who were treated with dexamethasone in the hospital but didn't [00:22:30] recover. I advised the doctors to switch to either methylprednisolone or prednisone and we saw a difference. So if I had to give you my opinion on what is best I would say methylprednisolone and prednisone which are far more potent than dexamethasone, easier to administer and of course cheaper. And of course a little less problems with hypoglycemia. I've had patients who were treated with dexamethasone in the hospital but didn't [00:22:30] recover. I advised the doctors to switch to either methylprednisolone or prednisone and we saw a difference. So if I had to give you my opinion on what is best I would say methylprednisolone and prednisone which are far more potent than dexamethasone, easier to administer and of course cheaper. And of course a little less problems with hypoglycemia. I've had patients who were treated with dexamethasone in the hospital but didn't [00:22:30] recover. I advised the doctors to switch to either methylprednisolone or prednisone and we saw a difference. So if I had to give you my opinion on what is best I would say methylprednisolone and prednisone which are far more potent than dexamethasone, easier to administer and of course cheaper.

Philip: [00:22:50] Absolutely, definitely. I'll summarize and remind people of something very important that you did [00:23:00]. Part of your strategy was reconnaissance. You educated your patients on what to expect and that was one of the reasons your strategy was so successful. What exactly did you tell your patients?

Shankara:[00:23:22] Philip, this education continues to this day, I think it's the most important part of saving lives. I realized that [00:23:30] things take a turn on day eight. And in order to determine this eighth day, it is crucial to find the first day of the disease, the day the disease began. This sometimes confuses our patients. If you ask a patient how long they have been feeling unwell, they will say, “My cough started that day or my fever started that day. However, it is important to question these symptoms in order to determine the exact timing of the onset of the disease. So my question would normally be: Were you fine the day before [00:24:00] and then the patient says no, I started to cough but the day before I had a

fever. And were you really well the day before? By asking, you can pretty much figure out the first day. Based on this, I informed each patient about what could happen a week later. Patients need to understand that what happens on this eighth day is crucial. So my initial treatment was to get her rid of the viral disease, which [00:24:30] wasn't a big deal. We all had viral diseases, flu, and respiratory illnesses, so I let them do the usual. I've made sure they get past that initial phase before day eight. We're doing our best so that by day eight you'll be ready to fight a war if you have to. And so everyone was informed that on the eighth day it is better to be healthy, that one should do everything possible to quickly overcome the initial illness. If [00:25:00] symptoms worsen, come back in time so we can adjust your treatment. And on the eighth day, if you have any new symptoms, I need to know right away. The first wave was shortness of breath. I mean, if a patient gets short of breath, they go to the doctor. Everyone knew that. However, with the second wave, I found that the first symptom on day eight was not shortness of breath, but rather fatigue. So I have to explain to the patients that on day 8 they were fine [00:25:30] and on day 8 they woke up with an overwhelming feeling of tiredness. That's enough to get me back to the practice. And of course they understood the seriousness of the situation. I believe, there was enough fear in the world that they understood that they had to come back to me on the eighth day. I didn't need to scare them any more about what was about to happen. And so they reported back in time. You see, Philip, I think the problem with that is that, unlike other allergic reactions, which are obvious, it's not that obvious. If you have another allergic reaction, you know something is wrong and you go to the doctor. Here, with Covid, it was [00:26:00] an allergic reaction set off in your lungs. And of course the lungs have a remaining capacity of two-thirds. So it can take a while before you realize how critically ill you actually are. And that loss of time en route to treatment makes it that much harder to contain the process. We know that when dealing with severe anaphylactic, anaphylactoid reactions, the speed and aggressiveness with which they are addressed is critical. And so I had to [00:26:30] make sure the patients who had that reaction got back in time. For this purpose, the clarification of the eighth day was essential. I had support from some priests in my neighborhood. Unfortunately, they developed COVID, and we did this day eight educational talk. All three recovered [in the viral phase], then had an aggravation on day eight and rushed back to me. And of course I was given the opportunity to start aggressive [00:27:00] treatment early and all three recovered. I think if you predict the day someone will start dying it's almost biblical. And since they were priests, they looked at me and said, man, that guy must be a holy man, because he told us when we were going to start dying. And so they went back into their communities and started educating them about this phenomenon. So the eighth day in my region became a topic of conversation very early on. That was a good thing, because the people around me understood that the eighth [00:27:30] day is crucial. I had uneducated patients, real illiterates, sitting and waiting for me in the tent you saw. And I heard them tell other patients about the importance of the eighth day and not to ignore it. So the news spread very early among the population. And since they were priests, they looked at me and said, man, that guy must be a holy man, because he told us when we were going to start dying. And so they went back into their communities and started educating them about this phenomenon. So the eighth day in my region became a topic of conversation very early on. 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Philip: [00:27:48] Absolutely. Well, so Shankara, like I said, I spoke to you in April and then your story spread like a virus and a lot of people heard about you. [00:28:00] In a weird way, similar to what's happening here with social media, people are making their doctors aware of what you're doing. It's strange that it's not the doctors who have been briefed about you, it's the public saying we need to know what you're doing in order for us to be protected. But there were also countries that approached you, right?

Shankara:[00:28:26] Yes, Philip, at the very beginning [00:28:30] after the interview, the first one. It was MTAM, the Malaysian Translational Medical Association, who contacted me and they wanted me to come and train doctors more online during lockdown. And so we organized a meeting where I could educate the doctors about the perspective, the presentation of the disease, and how to initiate treatment. 183 doctors were present at this first meeting. After that, the Association [00:29:00] broke this meeting, which lasted I think three and a half hours, into parts and posted it on their website as a training course for doctors around the world, so that every doctor could log in and understand the perspective could. This gave me the opportunity to reach a wider audience, even without my presence. And a few weeks later, I got a call from the health minister in Meghalaya, a state in northern India, and he wanted me to [00:29:30] train the doctors there. We had this first meeting. He invited all department heads to a session to understand this new perspective. So the first meeting was with the heads of ….. and the ICUs. So I explained to them how to represent the disease and how we should see it. Two weeks later they called me again and said to please come back and train the rural doctors in Meghalaya [00:30:00]. And the ICU specialist I spoke to two weeks earlier wanted to give a testimony before the training started. And he said that my view after he v a state in northern India, and he wanted me to [00:29:30] train the doctors there. We had this first meeting. He invited all department heads to a session to understand this new perspective. So the first meeting was with the heads of ….. and the ICUs. So I explained to them how to represent the disease and how we should see it. Two weeks later they called me again and said to please come back and train the rural doctors in Meghalaya [00:30:00]. And the ICU specialist I spoke to two weeks earlier wanted to give a testimony before the training started. And he said that my view after he v a state in northern India, and he wanted me to [00:29:30] train the doctors there. We had this first meeting. He invited all department heads to a session to understand this new perspective. So the first meeting was with the heads of ….. and the ICUs. So I explained to them how to represent the disease and how we should see it. Two weeks later they called me again and said to please come back and train the rural doctors in Meghalaya [00:30:00]. And the ICU specialist I spoke to two weeks earlier wanted to give a testimony before the training started. And he said that my view after he v We had this first meeting. He invited all department heads to a session to understand this new perspective. So the first meeting was with the heads of ….. and the ICUs. So I explained to them how to represent the disease and how we should see it. Two weeks later they called me again and said to please come back and train the rural doctors in Meghalaya [00:30:00]. And the ICU specialist I spoke to two weeks earlier wanted to give a testimony before the training started. And he said that my view after he v We had this first meeting. He invited all department heads to a session to understand this new perspective. So the first meeting was with the heads of ….. and the ICUs. So I explained to them how to represent the disease and how we should see it. Two weeks later they called me again and said to please come back and train the rural doctors in Meghalaya [00:30:00]. And the ICU specialist I spoke to two weeks earlier wanted to give a testimony before the training started. And he said that my view after he v how to represent the disease and how we should see it. Two weeks later they called me again and said to please come back and train the rural doctors in Meghalaya [00:30:00]. And the ICU specialist I spoke to two weeks earlier wanted to give a testimony before the training started. And he said that my view after he v how to represent the disease and how we should see it. Two weeks later they called me again and said to please come back and train the rural doctors in Meghalaya [00:30:00]. And the ICU specialist I spoke to two weeks earlier wanted to give a testimony before the training started. And he said that my view after he vbought, was introduced in his intensive

care unit. Just by changing the way he approaches patients in his ICU and understanding that we are dealing with hypersensitivity pneumonitis rather than COVID pneumonia, he managed to save three [00:30:30] out of four intubations to avert his intensive care unit. He found that remarkable. And he also said that his grandmother was admitted, she was 84 years old. She had come in 60 percent saturation on day 13, if I remember correctly. They were all very concerned about her because she was diabetic, but he did what I taught him to do. The day I made my presentation, it was her fifth day in the hospital. Her saturation was now up to 98 [00:31:00] percent. Her biomarkers had all stabilized and the results of the latest blood work were awaited and he was confident that she could be discharged the next day. And I think when he realized how easy it could be to reverse the problem, he decided that he shouldn't be allowing patients in this critical condition to come into his ICU and that this information would be shared with doctors in the country so they can prevent this and prevent patients from coming to the hospital in the first place [00:31:30]. And so I was invited to train all the country doctors. That has changed, I trained doctors in Sri Lanka. I have now also conducted a workshop in Singapore to train doctors there. I have been contacted by various doctors around the world asking for information on how to do this. However, the first group of doctors was in India. Before our interview, Philip, I shared this with the chat group of my colleagues, my senior year students, [00:32:00] my classmates. For her it was a revelation. We spent three weeks discussing the perspective and specifics of what I found. And so it was used in India very early on. Long before any other country knew about it. Many of my colleagues in India who use this have also shared their experiences with us. I think it's not about numbers, it's not about how many patient lives are saved. I can't do everything with my two hands. [00:32:30] And as more people gather around me and understand and adopt the perspective, the more lives we can save. That's what interests me. So I tried to stay out of the administrative structures. I knew there would be stumbling blocks along the way. So the goal was always to educate the doctors about my point of view and to inform the patients. And I think if those two parts of the community understand that, I don't have to ask anyone's permission to save someone's life. So that was the basis of my [00:33:00] push from the moment we had our conversation. That's what interests me. So I tried to stay out of the administrative structures. I knew there would be stumbling blocks along the way. So the goal was always to educate the doctors about my point of view and to inform the patients. And I think if those two parts of the community understand that, I don't have to ask anyone's permission to save someone's life. So that was the basis of my [00:33:00] push from the moment we had our conversation. That's what interests me. So I tried to stay out of the administrative structures. I knew there would be stumbling blocks along the way. So the goal was always to educate the doctors about my point of view and to inform the patients. And I think if those two parts of the community understand that, I don't have to ask anyone's permission to save someone's life. So that was the basis of my [00:33:00] push from the moment we had our conversation. if these two parts of the community understand that, I don't have to ask anyone's permission to save someone's life. So that was the basis of my [00:33:00] push from the moment we had our conversation. if these two parts of the community understand that, I don't have to ask anyone's permission to save someone's life. So that was the basis of my [00:33:00] push from the moment we had our conversation.

Philip: [00:33:02] And you've had some success with colleagues in South Africa too, haven't you?

Shankara:[00:33:08] Yes, in South Africa. Unfortunately, South Africa was the last country to respond to my work. A colleague in Durban, a doctor I didn't know. He learned about my work from the international recognition it received. I think he figured he had to check it out. [00:33:30] He did this almost in disbelief. He is an ICU-trained doctor who was present during the first two waves of COVID, but on an outpatient basis when hospitals were too busy or patients refused to go to the hospital, he was called in and he used the World Health Organization protocols and tried all the other little protocols that were out there for anticoagulation and the rest. And when he found out about my protocol, he tried to contact me. By now it had gotten to a point where I [00:34:00] feared I was being lured into a trap because there was this eerie silence here in South Africa. That's why my wife deleted his emails. She said: "Who is the doctor asking you to treat the patient? You don't go and give him advice so he blames you for everything he didn't do right. And then he got my cell phone number and he contacted me and we talked and he realized I was right about the treatment for the first four patients. He had admitted the first two patients to the hospital. I think he was too skeptical to try it. And then on the next two he decided to try this method and the very next day they were feeling much better. They had improved drastically. So he went to the hospital for the two patients he had admitted and insisted they change treatment and they were discharged after two days. So he felt he had to discuss this with me. And I think it's a simple view when you're treating a hypersensitivity or an allergic reaction. So I [00:35:00] explained the point of view to him. I filled him in on all the little niceties we had and he carried that on. And now, during the third wave, he has treated over a thousand patients. At the end of the third wave, we took the opportunity to look back and see where we've gotten on this journey. In the first and second waves - remember this is a doctor treating critically ill patients - he had an average of one to two [00:35:30] deaths per 10 patients. In the third wave,

he had treated over a thousand patients and not recorded a single death. He was so grateful to me for reducing mortality in his practice. When I met him, he was traumatized. He had a co-worker who passed away, he had an intern who passed away, and he's so thankful he has no new deaths in his practice. But of course it could also be something else. It could be a new variant. [00:36:00] All of these things are spoken of in disbelief by people. And so one day I sat with him and said, "Look, the speed of recovery is what I look for. Let's take two patients who are similarly ill. And let's look at the different treatment methods and how quickly they recover. So we took a patient with a 70 percent saturation as the baseline. And he said: "In the first and second waves he was on steroids, tocilizumab and remdesivir. [00:36:30] And it took about three days on average for the patient's condition to improve from 70 percent to 75 percent. And many patients went the other way and needed ventilation. In the third wave, he began using this perspective with antihistamines and montelukast and a high-dose steroid to stem the reaction. He realized, that in patients with 70 percent saturation, saturation improved to 85 percent within four hours. And after another four to six hours, they [00:37:00] had improved to over 90 percent. So I laughed and thought I explained it to him. I said, “Well, now you understand why I denied the need for oxygen. Reversing hypoxia was timeless enough that I didn't have to invest in oxygen. We laughed because not long ago he sent me a message on WhatsApp saying, 'You know, in the first wave, everyone was talking about oxygen. So I ran out and spent the first wave chasing oxygen. In the second wave, it was remdesivir. And I ran out and spent the [00:37: 30] second wave chasing remdesivir. In preparation for the third, I invested R100,000 in oxygen and R100,000 in remdesivir. And he said, "And then I met you. And now, at the end of the third wave, I have 100,000 Rands of both lying unused in the corner of my office. I need to find a doctor who still treats the problem the old way to dump it on him.

Philip: [00:37:52] But seriously, you know, Shankara, I think part of the problem is that people didn't listen to you. Sometimes they hear [00:38:00] that one doctor has treated so many patients and there have been no deaths and then they just say it's impossible and they haven't listened because anyone listening to you knows that you know what you are talking about. So can you explain how it is that the western world in particular, with all their financial power, doesn't come knocking on your door and trying to figure out how on earth [00:38:30] you can do that? Why is that?

Shankara:[00:38:34] Philip, I think the western world is too fixated on protocols. She's too fixated on following a particular methodology when treating people. And I think that's where the problem lies. You have to examine every patient. Every patient is unique. And I think that's where the problem lies. Also, I think the authorities have mandated too many [00:39:00] protocols in the past. And so doctors got used to just sticking to those guidelines and following them. I also think there has been a decoupling. Right at the beginning of my career, I was offered a position as an intern in many different specialties due to my scientific background, and many of my colleagues felt it was a waste of my knowledge that I chose to remain a family doctor. But [00:39:30] I felt that the broad base of understanding was more important to me. I've seen an ophthalmologist forget how to take a patient's blood pressure because he hasn't done it for a long time. So I wanted to keep this broad basis for my understanding. And so I practice medicine. So I understand that the ordinary GP finds that daunting at times because GPs tend to be a sorting station and when things get too complicated we send them to [00:40:00] specialists and then the specialists sit in Generally in a hospital without really understanding the patient and family dynamics. I think all of that matters, and I think we need to get back to basic medicine, to understanding the patient that's in front of us, and to science. I still have a great passion for science and nature and an understanding of these things. You know, I still fix my own bikes, [00:40:30] and I'm a very mechanical person, so yeah, I've stuck my finger in a socket too many times and gotten a shock. So I know science is my playground and I think it's that passion that shines through in understanding this disease.

Philip:[00:40:45] So listen, I laid the foundation because I wanted people to understand that you know what you're talking about and that your insights are extremely valuable. That leads us straight to [00:41:00] the question about omicron. Because here we may be at the beginning of a pandemic again if this virus bypasses all vaccines, which is possible. We're technically right at the beginning, two years ago, almost December, that's when the pandemic started in December 2019. It's now December 2021 and could potentially be all the way back. [00:41:30] The first question is: What do you think about the events in South Africa? I mean, it's still very early and you're almost, how do you say? You have been punished for your research by identifying the variant. However, it seems that most cases occur in South Africa. So one could assume that this is the probable place of origin. What do you think?

Shankara:[00:41:59] Oh Philip, [00:42:00] I think when you look at the transmission of a virus and the number of cases, we need to look at how we get there. Other countries have lockdowns that are strictly enforced. This is not the case in South Africa. So there is a possibility of further dissemination. Of course we have a faulty PCR test. Therefore, the countries that

test all residents are likely to find more cases. So I think all of these [00:42:30] little nuances play a part. So we can't really say that Omkcron originated in South Africa. We could have more cases. I think one of the first cases was discovered in Botswana. And then a case in Hong Kong. So yes, it is an airborne virus. You never know. Yes, we have identified it. To understand if we're dealing with something new, we need to understand the science behind it and know what's in store. But who found it [00:43:00] is really irrelevant. There are no limits to the spread of air. So I think it's much more important to understand what omicron is and how it's changed, how it's mutated, how it presents itself and where we're going with it. The fact that South Africa has identified it shows that we have a very good infrastructure here for genetic sequencing and understanding of infectious diseases. We in Africa are very susceptible to this. We have a large proportion of the population that has [00:43:30] HIV. We are prone to other diseases, infectious diseases. So our infrastructure for understanding infectious diseases is quite advanced. So I didn't expect that we would be admonished for our progressiveness. So yes it is

Source: https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(21)02758-6/fulltext 7-day average of infected in cases / 100,000 and days since wave began. As seen in the red circle, the omicron variance curve rises earlier and faster. Omicron is therefore much more contagious than all previous variants. You are in the red circle

Philip: [00:43:44] That's exactly what happened. I have a piece of work here that was recently published in The Lancet. I'm trying to remember who wrote the post. Well, it was actually just a comment. And here [00:44:00] is this chart comparing the other variants, alpha and delta, with delta being the one that has the highest peak. But here you have Omicron, and you can immediately see that the curve here is quite different from the other variants.

So we must [00:44:30] adjust to the fact that this disease is far more transmissible than any other and therefore unlikely to be stopped even if we try to limit its spread globally. So we have to be prepared for the virus to spread. Do you have an opinion?

Shankara:[00:44:55] In the general evolution of a pandemic, Philip, the variants [00:45:00] that evolve over time tend to cause less disease, milder symptoms, but they tend to to become more contagious. The goal of a virus is to spread. I don't think a dead host will do him any good. So the disease tends to become milder, but the contagiousness of the virus itself increases over time. So there will be natural selection for contagious [00:45:30] variants because they spread faster through the population. That would be the natural course of a pandemic. So it starts with all the fear and serious illness. Then it slowly becomes endemic, that is, it becomes a mild, transient but highly contagious disease, which is spreading quickly. So we're dealing with a far more contagious variant, but for now we're dealing with a [00:46:00] milder disease that still has a lot to learn about. As for the public health measures, I didn't see any benefit from them from the start. So if the lockdowns haven't worked, I don't think we can tighten the lockdowns any further, do we? So even a contagious variant will not benefit from nonsensical health measures. Because it is a respiratory virus, it is transmitted through the air and through aerosols. [00:46:30] We need to consider all of these facts in our public health strategy. So I think one should limit large gatherings and close contact, and that you should make sure that people understand the hygiene regulations and all those things. But tough lockdowns, travel restrictions and all those things aren't going to make much of a difference in my opinion, but we're definitely dealing with a far more contagious variant. That doesn't mean it's a bad omen, though.

Philip:[00:46:58] Hmm. That's a very good argument. I [00:47:00] think that - and I'm going to mention some of my research on autoimmunity here - similar to the hypersensitivity autoimmunity you mentioned, they are very similar concepts as far as the trigger goes . And what I mean to say is that the virus itself or the viral infection was never really the main

problem, as we can see until day eight when the viral load peaked. People are generally fine. If [00:47:30] her condition worsens, it's because of the immune response to the virus. This part is a bit more unpredictable. The question now is whether we have a transmissible virus. Will there be more cases of hyperimmune activation, what is technically the cause of death in COVID 19? It is never a viral pneumonia. It's about the body's response to [00:48:00] the virus.

Shankara:[00:48:03] Yes Philip, as I saw with the second wave, we had a mutation in the spike protein that caused it to be much more allergenic. And because of this increase in allergenicity, there has been a far greater increase in mortality and morbidity. Thus, the pathogenicity of the variant was determined by its ability to [00:48:30] elicit an inappropriate response from the host, and the severity of that response determined the severity or mortality and morbidity of that particular variant. So now we have another mutation in the spike protein. And of course we are dealing with a mild illness here, and of course that fits in perfectly. We probably now have spike proteins that are less allergenic and therefore cause milder diseases. And you won't see the mortality and morbidity because you don't get the same [00:49:00] inappropriate immune response on day eight. However, it may also be that despite being a less allergenic spike protein, people have developed a certain level of tolerance to this protein. And that's where she's going to play a role. So the spike protein is the primary causative agent of COVID disease, not the coronavirus itself. The coronavirus is just a vector to expose you to the spike protein. So the hunt for the virus is really nonsensical. [00:49:30] have developed some degree of tolerance to this protein. And that's where she's going to play a role. So the spike protein is the primary causative agent of COVID disease, not the coronavirus itself. The coronavirus is just a vector to expose you to the spike protein. So the hunt for the virus is really nonsensical. [00:49:30] have developed some degree of tolerance to this protein. And that's where she's going to play a role. So the spike protein is the primary causative agent of COVID disease, not the coronavirus itself. The coronavirus is just a vector to expose you to the spike protein. So the hunt for the virus is really nonsensical. [00:49:30]

Philip: [00:49:30] Yes. So an interesting question from Ramon is whether your patients' symptoms differ depending on their vaccination status.

Shankara:[00:49:44] Philip, this is something that I had to look at very carefully, and I found that I could divide these patients into different subgroups. On the eighth day, we're dealing with a disease caused by the spike protein. And [00:50:00] that's why I classified this as spike protein disease, because we see that in a lot of different parts of the body after vaccination. There are many different places where I see spike protein causing problems, so I decided to call that spike protein disease. When the mRNA vaccines were introduced in South Africa, I found that many patients came to me seven to ten days after vaccination [00:50:30] with symptoms that looked like COVID. Many of these patients subsequently tested positive for COVID. This seemed very unusual and there were too many of them to be just a coincidence. In these patients, I found that in some the disease suddenly worsened after three or four days, which was not the case before vaccination [00:51:00]. And only in those patients who had received the vaccine, who presented a week to 10 days later, the situation could deteriorate very quickly. So I realized that in some patients I was not dealing with a viral disease. I'm dealing with spike protein disease. So the vaccine caused the body to produce peak proteins, and on a given day those proteins triggered disease similar to what I saw on the eighth day. A patient who had been ill for three days suddenly showed signs of deterioration with hypoxia. He wasn't really sick on the third day. His first day of sickness was actually the eighth day. He went straight to spike protein disease and avoided a viral illness altogether. So I had to watch this subgroup of patients. I had to inquire about each patient's vaccination status and the exact date of vaccination so that I could carry out an appropriate risk stratification. So for any patient who showed up with these [00:52:00] symptoms just prior to vaccination, I did early biomarker testing to find out if it was actually an infection or just an allergic reaction to the spike protein acted, which produces the vaccine itself in her body. And with those who usually fell ill one month after the vaccination, I could assume that it was a breakthrough infection. I had given them enough time to show an allergic reaction [00:52:30] and if not, we had also given them enough time to show an immune response to the vaccine itself. So these were breakthrough infections. And the breakthrough, the real breakthrough infections after vaccination have shown up in exactly the same way. They had the viral phase, which got a little worse by day eight and had exactly the same symptoms. Yes, those who had such a breakthrough infection one to two months after vaccination had milder symptoms, [00:53: 00] and many of them survived the second phase of the disease really easily with one treatment. But I think that has little to do with the immune response that this vaccine actually elicits, because if it did elicit an immune response, they wouldn't have gotten the infection in the first place. I think it has more to do with being exposed to the spike protein and developing some tolerance to that protein. So if you're exposed to a spike [00:53:30] protein on day eight because you've been desensitized, you don't react as violently. And of course we have seen that the claimed reductions in the severity of disease and death from the vaccines tend to wear off even three or four months after vaccination. so i think when the vaccine stops producing

spike proteins and you become more exposed to it, the level of tolerance it gave you begins to erode. So if you get infected after a period of time [00:54:00] you don't have that benefit of desensitization anymore. So you have a severe reaction to the virus again. Thus, repeated vaccinations to reduce serious illness and death work through desensitization or tolerance building rather than an immune-mediated benefit that prevents one from getting the infection in the first place. It's the therapeutic benefit that [00:54:30] I see. you no longer have this benefit of desensitization. So you have a severe reaction to the virus again. Thus, repeated vaccinations to reduce serious illness and death work through desensitization or tolerance building rather than an immune-mediated benefit that prevents one from getting the infection in the first place. It's the therapeutic benefit that [00:54:30] I see. you no longer have this benefit of desensitization. So you have a severe reaction to the virus again. Thus, repeated vaccinations to reduce serious illness and death work through desensitization or tolerance building rather than an immune-mediated benefit that prevents one from getting the infection in the first place. It's the therapeutic benefit that [00:54:30] I see.

Philip:[00:54:31] Yes, interesting. Interesting. One of the other considerations I've done some research on is interferon. For those who don't know, interferon is probably our main defense against the virus because it stops the cells in the body from making proteins that the virus needs to multiply. So when interferon goes up, it blocks [00: 55:00] the virus multiplies and the rest of the immune system can pick up the infected cells. One of my questions is when people have had their booster shot and had a very strong immune response. The interferon must be suppressed. What if interferon is suppressed and you then become infected? I wonder [00:55:30] if that makes it harder for the respiratory system to clear the virus, if it already eludes the antibodies of the vaccine? Is there a longer-lasting infection? And if you do that, what impact will it have? I'm trying to think about it, and I think it definitely needs your observation to see if there are any patterns that emerge because I [00:56:00] expect it's going to play out differently. Either it disappears, as we hope, or it will appear in a different way. that it will turn out differently. Either it disappears, as we hope, or it will appear in a different way. that it will turn out differently. Either it disappears, as we hope, or it will appear in a different way.

Shankara:[00:56:11] Exactly, Philip. You see, I trust natural immunity. I have no reason not to. Natural immunity exhibits this great diversity. The mechanism is that you chop this virus into 100 different parts and develop an immune response to each one of them. So you have a wide [00:56:30] range of antibodies to fight the virus in a natural infection if you're unvaccinated. The problem is that with a vaccine, especially a vaccine that is very specific for the wild-type spike protein, you elicit a very narrow range of antibody responses, and the antibody response is against a spike protein that no longer exists . So we are dealing with non-neutralizing antibodies or non-sterilizing immunity. And [00:57:00] yes, that will bring its own problems. First, breakout variants and so on could emerge. My concern, however, is that a vaccinated patient, when naturally infected, might not mount the same robust, broad, and diverse natural immune response to the virus as an unvaccinated person. So vaccinated patients [00:57:30] might be vulnerable to reinfection because they haven't developed a full arsenal of antibodies to fight off a second infection. Because of this, I think we need to be very careful about collecting the data. People, who have received two vaccinations and are past the expiry date of those vaccinations cannot be classified as unvaccinated. If they are classified as unvaccinated, then they will [00:58:00] dilute or disrupt this data pool of natural infection in the unvaccinated. So patients who have been vaccinated and want a booster shot must be classified as something else because they received the vaccine and suddenly two doses later, six months later, these patients are labeled as unvaccinated despite having been vaccinated and long term effects of the vaccine will have. So we are dealing with four subgroups of patients [00:58:30]. There are those who are immunologically naïve and have never had an infection. We have those who have had a [symptomatic] infection and have developed a good immune response. In the group of the vaccinated are those who are naive and have never been in contact with the virus and have been vaccinated. And we have those who responded to natural infection and then got vaccinated. So there are four different subsets of patients that need to be studied to understand what the vaccine actually does. Is it [00:59:00] that the vaccine damages the natural immune response that you got from the virus? You developed a good natural immune response to the virus and then got vaccinated. Would that affect the immunity you've developed against the disease? As long as we understand natural immunity and the role it plays,

Philip:[00:59:19] Yes, that's a very, very important point. I think part of the difficulty is that there's so much censorship right now that I'm still very careful about [00:59:30] talking about anything vaccine-related, even if it is i'm trying to think it through. But it doesn't allow you to really think and analyze what the results might be. We all hope everything is ok. But my perspective has always been that if things are not right, we need contingencies and we should try to [01:00:00] develop a strategy. And that's what I want to try to challenge your brain. Shankara, let's assume we get a pretty serious illness. What would you think first? What would you look for with your analytical mind?

Shankara:[01:00:21] Philip, it depends on the patient and their vaccination status. I think that this one will play the most important [01:00:30] role. We know about antibody-dependent amplification. We know the problems [mass] vaccination can bring in a pandemic. We know about the variants that could develop from this. I know the spike protein is the pathogen here. So I know that changes in the spike protein affect the severity of the disease. So I think the vaccination status of the patient is crucial to understand what is going to happen. [01:01:00] And I found very clear differences. Yes. Initially, we found that vaccinated patients became much less seriously ill and were hospitalized less often. But I understand, that this is the measure of tolerance, which decreases over time. So I think by exposing vaccinated and unvaccinated patients to different variants, we will be able to assess the robustness of immune stimulation, either by natural infections or by vaccines, and how that affects the [01:01:30] outcomes. So, the severity of the disease that we see must be measured against the patient's pre-existing immune status, whether from a previous natural infection or from vaccination. either through natural infections or through vaccines, and how this affects the [01:01:30] outcomes. So, the severity of the disease that we see must be measured against the patient's pre-existing immune status, whether from a previous natural infection or from vaccination. either through natural infections or through vaccines, and how this affects the [01:01:30] outcomes. So, the severity of the disease that we see must be measured against the patient's pre-existing immune status, whether from a previous natural infection or from vaccination.

Philip:[01:01:42] Hmm. Yes, it's quite a complex situation and I take a step back when I think about it. A key point that we're seeing around the world is that the Omicron people are trying to [01:02:00] get their booster shots, they're trying to get the vaccines. But however we look at it, the speed of spread of this virus is 30 to 60 days and how long will it take to build a robust immune response even at three weeks? So three weeks is 21 days plus 14 days more so that the immune system can become really active. So you're talking about thirty-five days. So all this is integrated into the picture. Also, the [01:02:30] interferon suppression I mentioned might have an impact. So it's a very complex scenario and my instinct tells me that now, two years later, we're in a more complicated situation than before. I think I lost Shankara temporarily and I hope he will come back. But yeah, it's an important point that I said: Are we in a more complicated [01:03:00] situation now that we have a much more contagious virus than two years ago when we had a virus that was contagious, but not as contagious as this one? Should we? What should we do now? Shall we repeat the same pattern as two years ago? Or is it an opportunity for us to look at things in a different way? Those [01:03:30] are all very, very important questions that we will and must try to answer. And it's not easy for health authorities to make decisions because there's so much we don't know. Honestly, even as part of Omicron, it's only nine days from November 25th to December 4th. And then from [01:04:00] the course that occurs in COVID 19, you can see a difference in terms of the way because there's a delay in the immune response. I think I managed to get Shankara back. I don't know what happened to you there. then you can see a difference in terms of the way because there is a delay in the immune response. I think I managed to get Shankara back. I don't know what happened to you there. then you can see a difference in terms of the way because there is a delay in the immune response. I think I managed to get Shankara back. I don't know what happened to you there.

Shankara: [01:04:19] We had a power outage. Philip, I think that's typical of my South African location.

Philip: [01:04:25] Yes. Yes / Yes. Well we're glad to have you back. We will? We are nearing the end [01:04:30] of our discussion, and there are two more things I want to share with you before we close. And one of them is that I wanted to draw your attention to your involvement in a very interesting organization, Doctors for the World. You are now a part of it and part of the leadership team of the project. What is it about [01:05:00] that interests you?

Skankara:[01:05:04] Uh, I think Philip, medical care around the world has diversified. Today the situation is quite different. So we have all these different actors that actually have an impact on healthcare and how we serve the population [01:05:30]. And I think there has to be some parity. The pharmaceutical industry has dictated funding, thereby determining the direction of research and the direction of treatment. And I think we need some grounding, some understanding that all the different areas of medicine need to be given equal weight and equal opportunity, because ultimately [01:06:00] is the patient that's in front of me , the representation of humanity. And whatever action we take, it must benefit the patient before me. And if they don't, then they don't benefit humanity. What appeals to me about the Doctors Federation for the World is that it offers us a wide range of languages, cultures, traditions, media, [01:06:30] governmental structures and their influence on healthcare. For example, the organization Doctors for the World has endeavored to bring together the great diversity of social classes in order to improve health care. And I think it's important not to be guided by who has the finances. I think Doctors for the World [01:07:00] is an interesting new development and I hope we get to a point where it

becomes an organization which will have an advisory role in all global health measures in the future. From an ethical point of view, I think that's exactly what we need to return to population-level medical interventions.

Philip:[01:07:26] Wonderful, wonderful. Yes, I agree. And for those who are interested in [01:07:30] Doctors for the World, we'll be sharing more information about it and raising awareness very soon. It is a collaboration of clinicians from all parts of the world in different languages who will learn from each other and share different techniques related to COVID and will also treat other diseases in the future. So it's a very exciting project that I'll be keeping people posted about. Finally, Shankara, what do you [01:08:00] think about where we are now? What I mean to say is that technically we are where we were two years ago, but in a worse position? Or are we in a better position now? What do you think?

Shankara:[01:08:18] Philip, I was asked this question at the beginning of the pandemic when I first published the article that was so controversial because it denied the need for vaccination because we could save lives with early treatment [01:08:30]. And someone asked me what I think the best vaccine is. I have said that the best vaccine would be to take the mildest strain of the coronavirus and make a live, weakened vaccine from it. Since it would be a live vaccine, we didn't need to vaccinate everyone. It would spread itself, almost creating an artificial pandemic to which everyone would develop natural immunity. This is how we could end the crisis. Of course, we went the mRNA or viral vector route with a very narrow immunity. When the omicroma variant comes into play, if it causes the mild illness we've seen and we don't experience any complications in the future because it's so contagious, it could do the same job as a live attenuated vaccine by spreading itself could spread. It would [01:09:30] infect large sections of the population with no death or disease. In this way, we could achieve the herd immunity we so desperately want. And of course the vaccines won't get us there. So in that sense there is hope, but I think we also have to be very careful. We have seen in the past that this virus behaves very strangely. And [01:10: 00] Of course we have to be very careful about the nature of the disease and the possible long-term consequences. So I think we should be cautiously optimistic. If anything, those countries that have far more deadly contagious variants should consider Omnikron a boon if everything proves right. [01:10:30] So closing the borders and excluding South Africa could be another unnecessary shot in the foot.

Philip: [01:10:39] Exactly right. Absolutely correct. Great way to end Shankara. I really appreciate your knowledge. I'm sure the audience will appreciate it. We hope that more and more clinicians, governments and organizations that can influence protocols and treatments will heed these words and most importantly [01:11:00] save lives. That is essentially our goal for the future: save lives. So thanks again Shankara. And I'm sure I'll be speaking to you again in the future. I wish you a nice evening. Just stay tuned while we make this outro, thank you very much. Thank you to everyone who was with us and I look forward to our next conversation. We will keep you informed.